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Editorial.

WHAT'S YOUR HURRY?

A GREAT surgeon once told his students: "A man may bleed to death from a severed carotid artery in three minutes. You can tie this artery in two minutes *if you are not in a hurry*." Is not the hidden meaning here that efficiency may only be secured without undue haste and that agitated hurry often leads to ineffectiveness and bungling? To think quickly, to act skilfully, to do effectively without being in a flurry or hurry are valuable and essential attributes of the successful surgeon and medical man no less than of the leader in business circles. The full carrying out of detail needs an intellect that works in orderly fashion. The mind must act with the hands; if the former gets much in front of the latter muddle will result, and if hands work in front of eye and mind we shall see unintelligible surgical hieroglyphics, and confusion will be apt to become worse confounded. In a world where unseemly hurry seems to be prevalent on every hand a word may be written in defence of a check on mere apparent speed. Most lasting and permanent things that are of value have been somewhat slowly created and constructed. The foundations of any safe structure are not jerry-built.

In connection with research we see accounts of conclusions quickly come to on subjects of which formerly it has taken years of inquiry and investigation just to scratch the surface. The findings are in direct opposition to those of previous labourers on the same subject in the like fields. Is it not probable that

research in a hurry is likely to be fallacious as regards its results? Steam was discovered before engines were built. If an engine, however beautiful, is constructed and the provision for the effect of steam overlooked the locomotive is not likely to go. If a fountain is built and the supply of water to it be forgotten there will be no perfectly formed or scintillating sprays of liquid. Supposing the object of the fountain to have been overlooked in the hurry to construct it, water brought to it in buckets at the last moment and forced through its jets will be a poor substitute for conduction pipes, make a bad scenic effect, and only lead to an inglorious and painfully interrupted spectacular display.

We are to-day seeing a picture of a country in a hurry, full of troubled energy without any definite action, knowing not what permanent effect it is out to produce, and regardless of the aftermath. It is thinking and acting like a confused man who is not master of his job, and no lasting good result will accrue to it for all its expenditure of effort.

Turning to another side of operations in a neighbouring region, what a splendid result for organized well-thought-out action we see in the activities of the Army Veterinary Corps. We are told by men who are no novices and who have seen things that the young veterinary surgeons at the Front "have indeed developed a real talent for quick and efficient surgery, even under fire." Attention to detail, good organization and dispatch without hurry have contributed to bring about the grand figures of 47,192 horses cured and rendered fit for military service again out of a total of 81,134 injured and incapacitated in the campaign.

Even in ordinary practice the call to a serious case of accident brings into play the faculty of quickness without undue haste and well-directed thought before action. The mind must run over everything that will be required to deal promptly and well with the emergency. If the needles be taken without the silk and the dressing without the needles, the hurry in getting to the case will merely be an example of misapplied effort. Energy is often mistaken by the ignorant for action. A message given by telephone in a hurry and without due thought often makes the summons only a call to the wind. Badly initiated and misdirected hurry makes slow progress in the wished-for direction and is liable to encounter pitfalls by the way.

In the little world of veterinary politics it is questionable whether undue haste has not occasionally militated against effectiveness. Productive technical, legal and political knowledge have to be gained by experience, and perhaps an intimate understanding of human nature is as important an element towards the achievement of success in these fields as it is in other walks of life where the human item is involved. Experience must always act as the perfecter of effective action. A man may be a poor pathologist, an indifferent clinician, a somewhat erratic surgeon, but given the will to advance and some years of work in his calling then his experience will always be worth listening to and taking the measure of. Complete theoretical knowledge tends to check the ineffectiveness of futile hurry and to produce instead co-ordinated action, all the parts of which work quickly, smoothly, and successfully together.

It is little satisfaction to us to hear practitioners, whether human or veterinary, glory in the number of cases they have visited in a day, the large aggregate of their operations as ordinary members of the profession, without being experts. We often wonder whether in their hurry many of their activities have not been useless or imperfect. The human machine can only accomplish a certain amount of work well in a given time. It cannot keep going at full speed perfectly and indefinitely. "One thing at a time and that done well is a very good rule as many can tell." In some neighbourhoods the best men are scarce, the general public accommodating and undiscerning, and there is a good time for him to whom Nature has given ne'er to be harassed, an unseeing eye, and a front unembarrassed.

There is something still to be said and written in favour of the good old motto, "Festina lente" (make haste slowly).

Many critics have given their views on our nation's operations in the Dardanelles. The futile hurry when the Fleet acted without the land forces has been condemned and its lack of success deplored. Those who think ahead may still believe that if that hurry had been successful we should almost certainly have been faced with similar distressing experiences to those of Lucknow and Khartoum.

G. M.

THE ANGLO-FRANCO-BELGIAN VETERINARY RELIEF FUND.

We can understand in a measured degree the position of our *confrères* in Belgium and Northern France who at one fell swoop have lost their homes and practices. It is characteristic of Britishers that they should not only be the friends of small nations, but also show practical help and sympathy with their allies and near neighbours in their misfortunes. A fund is to be started with the main object of repatriating and re-establishing in practice our unfortunate professional brethren. Notwithstanding that there are heavy calls on most of us in many directions at the present time, we hope that this fund will gradually grow so as to become a real help to those in need. No cause could appeal to each one of us more forcibly than this one and no object is worthy of more sympathetic and whole-hearted support.

LOCKJAW.

So far tetanus has been the greatest bugbear to the German surgeon, and Professor Czerny has stated that among 60,000 wounded Bavarians 420 developed tetanus and 240 died of this disease. In the military hospitals of Strassburg 14 per cent. of all the deaths were due to tetanus. It is believed that the great frequency of tetanus in this war is due to the trench fighting in heavily manured districts, where the infected soil often remains in contact with a wound for hours and even days before antiseptics can be applied. The fact, too, that the lacerated wounds caused by shell and shrapnel are relatively more common in this war than the wounds caused by rifle bullets may also partly explain the greater frequency of tetanus. The Germans have experimented extensively with anti-tetanic serum, but they are not convinced as to its merits. Some silly accusations have been made in the German medical press by surgeons who have noticed a greater frequency of tetanus among their own men than among the French, and who have accordingly declared that French bullets must have been intentionally infected.—*Manchester Guardian.*

General Articles.

HOG CHOLERA, AND THE VETERINARIAN'S RELATION TO SAME.*

By F. B. WHITFIELD, D.V.M.
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The above subject has been given me on which to write a paper for reading before this association. Though the name is a short one, gentlemen, there is embodied therein some of the most vexing and some of the hardest problems confronting the veterinarians of this State. It is a subject of the most vital importance to the farmers of our State in the live stock business, and also to you, the veterinarians of the State, who are depending on the farmer for a living. The more you see of this plague that is sapping the profits of a great industry from your people, keeping them handicapped in the production of food, the harder you should fight for its extermination.

Let us look into the cause of this dread, contagious disease which is annually destroying from fifty to sixty millions of dollars' worth of our animals, making empty, as it were, hundreds of thousands of pork barrels each year. We are told by the bacteriologist that this disease is caused by an ultramicroscopic organism which has never been seen by the most powerful microscope.

The causes which we are most interested in, however, are those which foster the spread of the disease, in other words, the predisposing causes. Among these we will mention the following: State and county fairs, streams, buzzards and other birds, hog buyers and sympathetic neighbours, and last, but not least, the patent medicine quack.

Many outbreaks can be traced to the fairs, for it is here that many hogs are brought together from all parts of the country, with little regard sometimes as to their condition or the health of the animals in the neighbourhood from which they came. An infected animal bought by the farmer from such a place may cause the loss of thousands of dollars' worth of hogs by being placed in a healthy herd. It is strange to see so many farmers, who seem to be intelligent men, make this mistake.

* Read before Alabama Veterinary Medical Association, Auburn, Ala., March 26 and 27, 1915.

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As to streams carrying this disease, I am sure that if you gentlemen could be with me in Houston County, where each man has a small stream running through his lots, you would be surprised at the number of cases that you would see transmitted in this way. Around a little town of Madrid in our county there are a great many of these streams, and on one occasion I went down there, during an outbreak of cholera in that community. Dead hogs could have been seen lying unburned out on the ground and in the streams, where the animals had gone to die. The stench in some localities was most repulsive, and the air was, we will say, black with buzzards. These products of decomposition were swept down the stream to infect herds many miles away. These same buzzards could fly with other birds to other sections of the country, and in this way infect the herds in the lots in which they would light to feed on other carrion. The dogs of the section in which such an outbreak occurred, by dragging the pieces of the dead animals from place to place, could scatter the infection.

Many cases of hog cholera are scattered by farmers borrowing the boar of their neighbour for breeding purposes. One of my clients, Mr. Crawford, of Dothan, loaned his fine boar to a neighbour for breeding purposes. This animal contracted the disease in two weeks. It is of utmost importance that we know from whence hogs come that we use for any purpose, and it is all the more important to know where our hogs go when we do loan them.

One of the hardest problems which the average veterinarian encounters in handling this disease is the way the farmers have of letting their animals run at large. By this method I am sure many herds are contaminated, for we well know that to handle any infectious disease it is highly necessary to quarantine any and all infected animals and the premises on which they are quartered, keeping other animals away from them. Many cases of hog cholera are started by neighbours who flock to the farmer's house and to his lot to offer any assistance, which they are always too eager to render in cases of this kind. They go into the lots, handle the animals, get the excreta from the sick ones on their feet, go back into their lots, and in this way scatter the infection among their well animals. The hog buyer in like manner carries the infection.

In no other case do I believe that the vendor of worthless medicines should receive heavier condemnation than is due that man who would, for the gain of a few dollars, deprive his fellow man of thousands of dollars' worth of property and food for his family. Gentlemen, I refer to the patent medicine quack. May his stay with us be short and be terminated as speedily as possible. Already it seems that we can hear his death-knell ringing, for with the men that the colleges of veterinary medicine are turning out each year in the field of service, Mr. Quack is going to the wall. He slips around guaranteeing cures with his wonderful hog cholera medicines, knocking the use of serum, and in this way prejudice against any treatment that may be good, is established by his failures.

Having determined some of the causes of this disease, let us briefly enumerate the symptoms. In the acute form we find that the following symptoms manifest themselves: There is marked rise in temperature, from 104° to 108° F. Loss of appetite and general depressed condition. The hog isolates itself from the herd and lies around burrowed in the straw and litter; exhibits a stiff, sluggish manner, being in a weak condition. First we may notice that the bowels are constipated, and later diarrhœa may set in, this diarrhœa being of an offensive odour A hacking cough may be present, and the animal's breathing may be considerably accelerated. The eyes may have a mattery discharge, and in some cases I have seen the animal seems to be affected with gastritis, as evidenced by the vomiting of glairy material from the stomach. He loses his appetite, and seems in some cases to be very thirsty as a result of the fever. In the chronic form the skin of the region of the belly assumes a parched appearance. These blotches may later be the foci for sloughing sores. I have seen these sores crust over in the hog that is recovering from the disease, these scabs falling off and leaving the skin covered by them denuded of hair. The urine may assume a dark colour as in other febrile diseases.

As to the period of incubation of this disease the time varies, but from observation it is safe to place the estimate at from three to twenty-one days.

In this, as in other disease, our most trustworthy method of diagnosis consists in making a careful *post-mortem* examination of the carcase. In *post-mortem* examinations we may find the

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following lesions: splotches or sores on the skin. These splotches may be found in most cases under the belly and between the legs. There may be a purulent conjunctivitis. Upon opening the body cavities we find the following lesions: The stomach presents a congested condition of the mucous lining, and small hæmorrhagic spots are seen. In the large intestine in the chronic form of the disease we find the characteristic button-shaped ulcers; a great many times near the ileo-cæcal opening; but in the acute form these are absent, and instead we see a congested mucous membrane. There may be a diffuse enteritis in the small intestine. The spleen in the cases that I have examined was in some cases congested, while in others there was very little change. The kidneys present in most cases small petechiæ, while in some cases this organ may be studded with red spots. The lymphatic glands are of a dark congested consistency, the black blood oozing from them on being cut into. The bladder may present cystitis. In the swine plague form the lungs are congested very much as in pneumonia. There may be small petechiæ around on the serous walls of the thorax.

Now comes the process of treating hogs in this disease. Here we may say, more than in any other disease with which we have to deal, that "an ounce of prevention is worth more than a *ton* of cure." This is perhaps the largest field of preventive medicine that the veterinarian of Alabama can be engaged in. As to the *Serum Simultaneous System* of vaccination, we can but repeat the words which, in a joking way, have been attributed to that famous old explorer, Balboa, who, as he looked out over the great Pacific Ocean, said, "Well, boys, it is a success." This preventive treatment has passed from the age of experiment and is now known to be a well-established and successful preventive.

As to the time that is suitable for the use of the simultaneous treatment for the prevention of hog cholera, I believe that it should be done as soon as the animals weigh 25 lb. to 50 lb. I believe that it is best to vaccinate all hogs in the herd as soon as they are large enough. In this way we are able to do it at the least cost, and thus the farmer can render his animals immune to the disease for all time. When the disease is in the neighbourhood it is imperative that the animals should all be inoculated. A great many farmers will put the work off until cholera gets into their herds and then come running to you for advice. In

this case, when the weather warrants it, I tell them to go home and kill all the hogs that they intend to use as food, for the use of the serum has not, in my practice, proved successful in that stage. One thing which is essential for the man's success in raising hogs, who does not vaccinate them as they become of the proper size, is to watch his neighbours' hogs as well as his own, for in this way he can determine when any animals around him are sick, and can then go to work with the serum treatment before it is too late. I would recommend that the early treatment be given all animals that a man is raising for breeding purposes, for in this way he can build up a reputation for raising animals that are immune and in this way build up his business.

In my work I have good results by using the *Serum Simultaneous* treatment in the following manner: The first essential to success is cleanliness always. I use a 30 c.c. syringe of the all-metal type for the serum and a small 2 c.c. syringe for the virus. The disinfectants I use are creolin and iodine. A small, ordinary drinking glass is used to hold the serum and an ordinary tin bucket is used for the creolin solution and cotton. The iodine is used on a cotton swab. A small pan with creolin solution is used to hold the syringes. With two good, strong assistants the animal is secured, being careful not to injure it, especially in the case of pregnant sows, for I believe that rough handling of pregnant sows is directly responsible for abortions which occur sometimes after treatment. Next I clean off the space in the axillary region of the limb with the creolin solution. Next the iodine is painted on the surface, which has been dried off with a piece of cotton. Next the serum is injected into the loose area in the axillary space, the dose being drawn into the syringe and measured by the graduated scale on the plunger. The needle is withdrawn and the skin pinched at the point of injection to prevent the escape of the fluid from the puncture. Next I inject the prescribed dose of virus after cleaning a point on the inner surface of the ham. The animal is now turned into a clean lot.

In herds where the disease is present I use only the *Serum Treatment* in those animals which show hypothermia. The others are given the *Serum Simultaneous Treatment*. My success in herds in which the disease has already broken out has not been very marked; nearly always 50 to 75 per cent. of them

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have died. Some of the bad luck I have had I attributed to impotent serum. My advice is to use the serum which has proved successful, even though some other firm may offer you special inducements in the form of commissions to use their products; for by the use of an unreliable serum you may place the use of serum in bad repute, and in this way injure the prospects for the eradication of the disease and be detrimental to your business as well.

Below I will enumerate some of the cases observed by myself:—

Herd No. 1, *A. D. Whiddon, Owner.*—These animals were given the serum simultaneous treatment because there was an outbreak of cholera near. Two of them were left unvaccinated. These two animals died, the others remaining healthy.

Herd No. 2, Mr Patterson, Owner.—In this case a neighbour's herd above his on a creek died of cholera. The infection, no doubt, was carried by the creek to his place. I vaccinated the herd belonging to Mr. Patterson. The animals in this herd all succumbed to this disease, except five out of the seventy vaccinated. The serum only was used in this case.

Herd No. 3, *Mr. Daughterty, Owner.*—These animals were on the same creek which watered the animals owned by Mr. Patterson. They were not vaccinated at all, and about 75 per cent. of them died.

Herd No. 4, *G. W. Pilcher, Owner.*—This herd, consisting of twenty-five head, were vaccinated before placing on peanuts left by hogs which had died from cholera. These animals have not contracted the disease yet, and the time when they were vaccinated was about two months ago.

Herd No. 5, *Mr. Crawford, Owner.*—These animals were infected by the loan of a boar which became infected and subsequently transferred the infection to the herd on being replaced after the neighbour had used him.

Herd No. 6, *Ed. Cannady, Owner.*—These animals were given the serum simultaneous treatment with the exception of one that was sick at the time of administering the treatment. He received a double dose of serum and recovered; the remainder did well.

Herd No. 7, *Mr. Sanders, Owner.*—These animals were sick at the time. Gave serum alone Lost one-half of them.

Herd No. 8, *Mr. Sanders, Owner.*—Gave these simultaneous

treatment. None sick. One of these died with cholera in two weeks.

Herd No. 9, Mr. Rogers, Owner.—Vaccinated 100 for him. None sick at time of vaccination. No bad news from them so far.

Herd No. 10, Mr. P. C. Bradshaw, Owner.—Cholera all around him. Twenty-five animals inoculated. All well.

The following is a table giving the dose of serum and virus used in this work, together with the weights of the animals:—

Weight of hog lb.	Serum c.c.	Virus c.c.
20	20	$\frac{1}{4}$
50	25	$\frac{1}{2}$
75	35	$\frac{1}{2}$
100	50	$\frac{1}{2}$
150	60	1
200	70	$1\frac{1}{4}$
250	80	$1\frac{1}{2}$
300	90	2
350	100	2
400	110	2

In giving the farmer advice on the subject of hog cholera it would be well to enumerate the following points: Keep your premises clean. Water your stock from your own well if possible, and if your well does not hold enough, dig it deeper, for we can dig wells or bore them with a lighter heart than in digging graves. Do not borrow your neighbours' animals. Keep people out of your lots and attend to your hogs yourself. Watch your neighbours' animals so that you can tell when your hogs will be in danger. If your animals die from cholera, do not go and buy more and place them in the same lot unless they are given the simultaneous treatment. Do not throw your hogs out on the open ground, but burn the hog and a stump at the same time, thereby killing two birds with one stone. Raise hogs that are worth the money spent in vaccinating them, and then you will not stop to consider the cost of serum treatment too long. Beware of hog cholera cures as sold by quacks. If you want information, go to your veterinarian at all times and ask him about different diseases of hogs If he is not interested in your welfare enough to talk to you, and does not seem to want to help you, cut him out and get another man to do your work. If your hogs have cholera tell your neighbours, so that they can profit by the information which you have given them. Feed cooked feed at all times. Vaccinate your hogs before they have an opportunity to contract the disease,

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and in this way ensure them against cholera. Do not buy hogs from around in the country unless you know what you are getting. When you do buy them, place them in a pen by themselves until twenty-one days have passed. Then, if they are still well, use them in your herd. This particularly refers to boars. Be careful to know that there is a rigid system of inspection at the fairs before you show any animals there. Bury all dead animals on your place and this way keep buzzards away. Study the method of vaccination and learn to do it yourself, having first learned under the directions of some veterinarian. Know that at all times success in the hog business, as in all others, is only attained by close attention to details and the study of your business.

Right here I may say, gentlemen, that one of the worst things with which we have to combat is the ignorance of the average farmer. We are vexed sometimes at the strange things they will do when we do not stop to think that most of them have no training in the prevention of infectious diseases. The average farmer in our State will talk politics for hours, and then, when he gets his man elected, knows nothing of the duties which he is expected to perform. Few indeed know that they hire a congressman to work for them as their public servant. He does not seem to know that to get the best information possible he has only to write for bulletins on work that this Government has spent thousands of dollars in hiring men to acquire the knowledge which may be gleaned from them.

Another thing which we have to fight to a finish is the patent medicines which are guaranteed under the pure food and drug laws of our country. Our great Government sees fit to permit this stamp placed on thousands of worthless nostrums, and I am sure that this very thing has done a great deal to keep our work along this line from being effective. Uncle Sam may spend his thousands in hiring experts, but as long as he permits such things as this he spends his money in vain; for with a farmer once fooled by some patent hog cholera medicine or serum, it is the hardest thing in the world to convince him that the serum made under the Government's direction is good. Fool him once, and you have a chronic grouch on your hands.

In fighting the disease first get the confidence of your man. Let him know that you have his interest at heart. Tell him the

cause of the disease, and point out the road he is to follow in combating it. Whenever you vaccinate animals for a client, try to have his friends there to see it done. Give them a little talk on the disease, and speak in terms that they can understand. When you are doing the work for an intelligent man, offer to show him how it is done, and under your directions let him do some of the work himself. Some here will say that this is taking money out of your own pockets, but stop and think what the financial condition of this country would be if every farmer had, say, twenty-five hogs to sell yearly made possible by this vaccination. I have men in Dothan whom I know, who have been inoculating their hogs for years, and as a result of this they are making money on them. You can rest assured that the man who sells his hogs will always have money with which to pay you for your trips, and you all know that the sweetest sound to a veterinarian's ear after the patient is out of danger is the rattle of the cash in his pockets.

The last Legislature of our State has seen fit to establish a hog cholera serum plant in Auburn. For this plant we are indebted to our State veterinarian. He has worked for years on this one thing, and at last it seems that his labours are to be crowned with success. We may stop and ask ourselves, "What does the establishment of this plant mean to the people?" It means this: More hogs and better hogs. More meat for our people and better fed people. The opening of another field of production for our farms. Less cotton and higher prices. Another product to be shipped and greater prosperity for our State.

In my opinion, and I believe I may add in the opinion of all veterinarians here present, this fight for the eradication of hog cholera is the most important one we are waging. The people of Alabama are large meat consumers and small producers. Hundreds of thousands of dollars' worth of western meats are shipped into our State which we could raise at home. The use of serum in eradicating this disease has proved a success in other States, and it will in ours. The present conditions demand that we raise more and more food products for our people. Gentlemen, the *American Hog* is one of the most important of these crops, and on him, to a great extent, hinges the prosperity of our people. It has been demonstrated that we can raise hogs

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as cheaply as in any section of this nation, and it is up to us, the veterinarians of this State, to render any and all the assistance possible to our greatest friends, and I may say the greatest people on this earth, the American farmer. Upon them you depend for your living, for, with the advent of the gas engine and automobile, your city business is fast falling away; as the horse in the city is doomed and those owned by our country friends are our only salvation. Therefore, let us keep these resources in as good condition as possible.—*American Veterinary Review.*

ANTHRAX IN ELEPHANTS.

By M. C. GUPTA.

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It is one of the most formidable diseases occurring in elephants. Considering the everyday difficulty in purchasing elephants for timber firms in Burma and the high price one has to pay to secure a really good beast for dragging timbers, it will not be out of place to discuss the subject. The mortality from this virulent disease amongst the elephants is still very high and now is the time to find out proper scientific methods to eradicate it as far as possible.

Definition.—An acute infectious disease caused by *Bacillus anthracis.*

Etiology.—The infectious agent is a non-motile, rod-shaped organism, *B. anthracis.* The bacilli themselves are readily destroyed, but the spores are very resistant, and survive after prolonged immersion in a 5 per cent. solution of carbolic acid, or withstand heating for some minutes in a temperature of 212° F. They are capable also of resisting gastric digestion. Outside the body the spores are in all probability very durable. The disease is conveyed sometimes by direct inoculation—more commonly by grazing in pastures contaminated by the germs. The spores may also be carried by the wind and in water. Local wounds and sores may also be infected by being brought into contact with the anthrax virus by the agency of flies. In the fodder the spores lie dormant for years.

The incubation period is generally from three to six days.

Symptoms.—(*A*) External, including (*a*) malignant pustule, and (*b*) œdema. (*B*) Internal: (*a*) intestinal, (*b*) pulmonary.

Malignant Pustule.—The pustule begins with an itching at a spot which soon becomes a livid red papule and vesiculates and forms a rough, black, central slough, usually surrounded by a zone of small vesicular pustules. It is early surrounded by an area of brawny inflammatory œdema, which varies greatly in extent according to the severity of the attack.

Œdema.—It appears soon after the animal shows the first symptoms, sometimes it is visible before any constitutional symptoms set in. Neighbouring glands are inflamed, especially in the places behind the parotid glands, between the limbs, groin, shoulder-joints, belly, or hind-quarters.

Briefly, the general symptoms are pains, dyspnœa, restlessness, with early prostration, paralysis with tottering gait. The attack is sudden with violent shivering or trembling. The temperature usually rises between 102° F. and 104° F. Frequent urination, urine fœtid. Sometimes intestinal colic attended with voiding of dark liquid or blood-stained evacuations. The symptoms in the *internal anthrax* are generally the same as described above, but without any swellings.

Diagnosis.—The malignant pustules, once one has seen them, give no difficulty in diagnosis, and if the pustule is not typical the intense brawny infiltration all round gives one the clue, and the bacilli are easily found under the microscope in a smear of the serum from a pustule.

The œdema or swelling increases in size rapidly and is hot, painful and doughy.

In any suspicious case the diagnosis may be rapidly made after the death with the microscope by cutting the ear and obtaining a blood smear, which shows the typical bacilli. But it should be remembered that the blood slides must be taken either an hour before or an hour after death. The anthrax bacilli invade the blood-stream shortly before death, and after death putrefactive organisms which resemble anthrax bacilli enter the blood-stream.

The *internal cases* are very puzzling, if one is not expecting them, and frequently cause sudden death. When seen in the collapsed stage they can hardly be diagnosed clinically. Fortunately these cases are few in comparison with external ones. The pulmonary form is very uncommon amongst elephants, and I have not come across a single case yet.

DIAGNOSIS OF ANTHRAX FROM BLOOD SAMPLES.

Owing to the importance of the subject, it will not perhaps be out of place to review a method of technique for the preparation, fixing, and staining of a blood film.

(*a*) *The Slide.*

In the first place, perfectly clean good glass slides are absolutely essential. These when produced and unsoiled must not be fingered and exposed to dust. They should be kept in a tightly stoppered bottle containing absolute alcohol, and when they are withdrawn for use, if not immediately required, they should be placed between two pieces of clean paper to protect them from the dirt and the dust which would otherwise rapidly reach them. Old slides may be cleaned by washing in soap and water, boiling for some hours in strong washing soda, re-washing in clean water, drying, bathing in strong sulphuric acid for three or four hours, washing gently and slowly, but thoroughly, in clean water, drying and stoving in absolute alcohol as above.

(*b*) *The Ear-prick.*

The ear of the elephant should be cleaned with soap and water to remove dirt which might otherwise give rise to crenated corpuscles, and then dried. A small droplet of blood should be produced and transferred to the slide.

(*c*) *The Spread of the Film.*

The technique of the smear is all important. It matters not what form of spreader be used, so long as the results obtained are good. An ordinary microscopic slide which has been chosen for the cleanliness and the smoothness of its edge will give perfectly satisfactory results. The result to aim at is the firm spreading at even pressure and uniform rate of a wide and continuous film to roughly cover two-thirds of the length and half the breadth of the glass slide with a smear of such a suitable thickness that a single layer of separated corpuscles is spread out.

The termination should not "tail," but should end in a regular line or curve (2, Plate I). The operation should be commenced as soon as possible after the blood drop has been placed on the slide. To give the slide the necessary support it should invariably rest on some firm basis.

Many of the mistakes in the preparation of blood films can be recognized at a glance. In some the blood has not been allowed to completely spread over its base before the film is made, whilst perhaps the film has been started slowly and finished quickly (3, Plate I). In others too small a blood drop has been taken, or too large a drop, whilst the spreader may have been applied with unequal pressure. Jerky spreading, due perhaps to anxiety to produce a good smear or to insufficient support to the slide, will result in a broken film (6). A greasy slide will cause the red corpuscles to form rouleaux, and a broken ragged film will result (7), whilst a good smear may be subsequently spoilt by flies feeding thereon (8).

When spread, films should be dried by waving the slide, held edgewise, backward and forward in the air. The films should then at once be folded in clean paper and sent in for examination.

Fixing the Film.

The next and most important step is to thoroughly fix the film. Either ethyl or methyl alcohol must be used; the latter is quicker but dearer. It may be conveniently purchased in B.W. and Co.'s glass capsules. Whichever form of alcohol be chosen, it must be without doubt absolute in quality. The least trace of water is harmful, and for this reason the greatest care must be taken to protect the fluid in stock. All bottles containing absolute alcohol should be thoroughly well stoppered. Fixation is best carried out in a bottle. Half-an-hour may at times be a convenient period, twenty-four hours is not too long. If there be any reason for urgent hurry ten minutes will suffice. The fixing bottle should be frequently refilled with fresh absolute alcohol. After fixing the film should be air dried as before, and once again protected from dust and insects if staining is not to be immediately proceeded with. The objection to drying with blotting-paper is that foreign matter is liable to be deposited on the smear.

Staining the Film.

Stain with Löffler's blue and some by Gram's method. Examine with the oil-immersion lens. Make a careful search over the films, looking on large cigarette-shaped bacilli, noting whether they are or not arranged in chains. Examine the Gram specimens and see whether the bacilli are to be seen in them also.

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A specimen of anthrax bacilli is appended in Plate II. The lower portion of the field shows bamboo-like chains and spores, and is taken from cultures. Methylene blue, x 1,000. The portion showing spores is stained with carbol fuchsin, decolorized by brief immersion in methylated spirit, and counterstained in methylene blue.

Post-mortem.

The *post-mortem*, however, always clears up the diagnosis; but, as a rule, elephants dead of anthrax must not be opened on any account. Plug all natural openings with disinfectants. If plenty of wood is available, burn the carcase whole, or bury in lime. The remainder of the burnt carcase should be thoroughly sprinkled with chlorinated lime and then buried properly. The top of the grave should be covered with a cart-load of ordinary lime. Great care should be taken to remove the tusks. The men employed to do this must not have any sores, abrasions, or scratches. Their hands should be properly rubbed with carbolic oil (1 in 20), and if by accident they cut their hands when extracting the tusk, it should be cleansed with a strong carbolic lotion (1 in 40) and dressed antiseptically. The men should be forbidden to continue work.

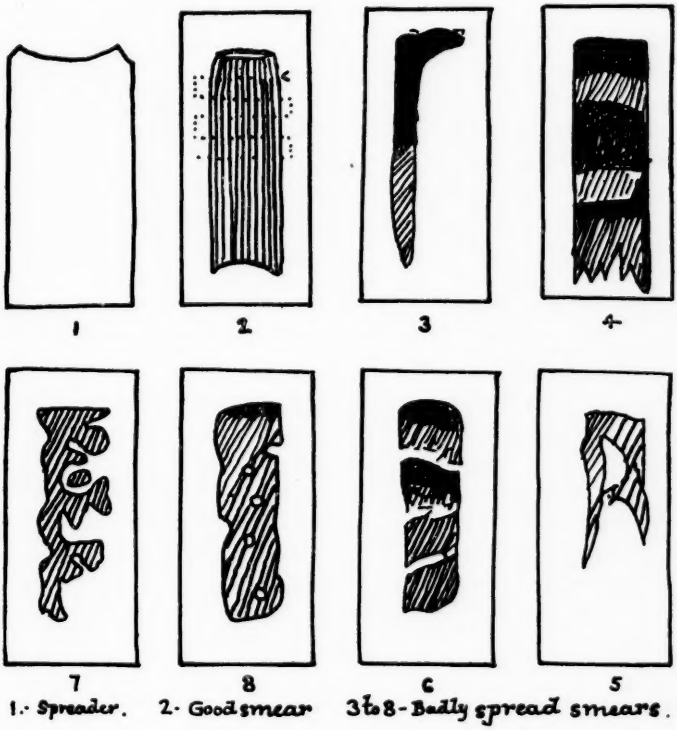
After the operation the men engaged should have a thorough phenyl bath, and all clothes worn by them during the time of operation must be burnt. All instruments, including dah, axe, knives and other things, must be put into flames and then washed in strong carbolic lotion. Any instruments once used in *post-mortem* examination should not be used in opening abscesses or other surgical operations. Care must be taken to restrict the infected area to the smallest limits. A deep pit should be dug alongside and the carcase tumbled in. Liberal quantity of lime should be sprinkled in the soiled area and a big fire made in this area. After this the area should be strongly fenced in.

Briefly, one finds on opening the abdomen a large quantity of free straw-coloured fluid, with sometimes flakes of lymph all over the coil of intestine. There is a peculiar jelly-like œdema of the mesentery, and the mesenteric glands are large, swollen, and hæmorrhagic. The peritoneum covering the bowel is congested and may show petechiæ, but the typical lesion appears to be numerous submucous hæmorrhages into the wall of the bowel,

which may be very extensive and destroy the mucous membrane, and are easily seen by holding up the bowel to the light, when they appear like black patches through the empty intestine.

The spleen is congested, dark, swollen, and soft, and may be diffluent. It swarms with bacilli. The heart-blood is thick and tarry, and the lungs and pleuræ are congested and may show petechial hæmorrhages.

PLATE I.



TREATMENT.

I generally adopt the following plan of treatment:—

In the Internal Cases.—Hypodermic injections of strong carbolic acid 2 minims per dose mixed with equal part of glycerine are recommended every four hours. If the animal is in a collapsed state hypodermic injections of 2 gr. strychnine tabloids

should follow the carbolic acid injections an hour after the first carbolic acid injection. These two injections should be given alternately until the grave symptoms disappear.

The suitable site for hypodermic injections which I generally prefer in the elephant's body is either the part close to the shoulder-joint or the haunch bone.

A fold of skin should be taken up with the finger and thumb of the left hand; the needle should then be passed through the skin obliquely under the surface. The syringe containing the injection should then be attached to the needle and the fluid injected slowly. If the injections cannot be carried on successfully 1 drachm of strong carbolic acid may be tried internally on tamarind every four hours, and 1 oz. dose of liquor arsenicalis may be given on tamarind every four hours, to begin an hour after a dose of carbolic acid.

N.B.—Syringes and needles should be boiled before and after use, and only water that has been filtered and boiled should be used for dissolving tablets.

External Cases—For malignant pustule, excision and actual cautery have their advocates, but the former is liable to be followed by a general infection, and the latter is difficult to carry out on elephants.

Sclavo's serum with or without excision may be recommended.

Another treatment recommended which has the advantage of being simple is cauterization with the solid caustic potash. I have tried this in many cases with very good results. It certainly seems worthy of trial, and is without the disadvantages of the treatment described above.

I generally follow the undernoted plan:—

In mild cases without fever, or cases seen after the fever has subsided and obviously clearing up, almost any treatment suffices, touching with pure carbolic and antiseptic precautions, or a crucial incision with the application of pure carbolic in the moderately severe cases.

In bad cases with high fever and much inflammatory œdema the following method gave excellent results, and no time should be lost in carrying it out. All around underneath the pustule 50 per cent. carbolic acid is injected with a hypodermic needle at close intervals. The idea of the treatment being that the pustule is shut off from the circulation by a zone of carbolized

slough, which seems to effectually prevent the infection becoming generalized.

MANAGEMENT OF THE OUTBREAK.

First and foremost is the *prophylaxis*, and it is obvious that the epidemic must be dealt with vigorously, when the cases will cease automatically, except perhaps for an occasional sporadic case. Any elephant dying suddenly without any apparent cause, whether "swelling" or "no swelling" on its body, must be taken as a suspicious case and its blood slide should be taken at once. If a little care is exercised in following up the method described above as regards taking the blood slides, no difficulty will be felt in examining the slides and coming to a correct

PLATE II.

Anthrax Bacilli.

diagnosis. Segregation should at once be carried out as far as practicable and particular care be taken to remove the elephants up stream from the infected camp, as spores are carried in water. The infected animal should be examined twice a day, and if any elephants develop any signs or symptoms of anthrax the camp should be changed at once to a place situated up stream, leaving the sick elephant on the spot. When examining infected elephants note whether eating usual fodder, any swelling on its body, body heat (temperature), and the particular look on the eyes of the elephants. If the following symptoms are absent from the beast on inspection it indicates illness.

The prominent signs of health are constant motion of trunk, swinging of the tail; mucous membrane of the mouth, vagina,

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ears and eyes are bright pink colour. The appetite is good; the animal sleeps but few hours at night, and immediately on waking commences to eat. As the anthrax spores lie dormant for years, proper inquiry should be made before locating a camp whether there was any epidemic amongst the cattle in the villages close to the grazing ground even years back (to be on the safe side four or five years).

Each elephant should be separated at least half a mile, and an attendant must be always with it to prevent the beast straying from a prescribed area. If there is no outbreak, within seven days the elephants may be removed to a non-infected area across a large stream.

All gear should be burnt, and iron fetters, &c., should be passed through the flame and kept unused for some time until the epidemic is completely over.

The tusks after being extracted should be properly soaked in strong disinfectants and put aside separately for at least a month. They should then be dried and chlorinated lime should be sprinkled on them.

REFERENCES.

[1] "Elephants and their Diseases," by Lieutenant-Colonel G. H. Evans.
[2] *Indian Medical Gazette*, April and September, 1915 numbers.

INTERVIEWED with respect to the continued high prices of meat in France, the Minister of the Interior (M. Maloy) said he was convinced that the arrival of foreign meat in appreciable quantities and in regular manner would bring about a fall in prices, but if in consequence of the attempts of speculators this result was not attained, the Government was very frankly determined to have recourse to the fixing of prices, as it had power to do by the law of 1791.

Abstracts.

THE CAUSE, PREVENTION AND TREATMENT OF RETAINED PLACENTA IN THE COW.

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When the cow has conceived, the segmented ovum has descended into the uterus and the embryo has formed, there are promptly thrown out the fœtal envelopes, through the medium of which the embryo is to receive its nutrition, to be protected from exterior influences and to be maintained in its position.

The embryonic sac develops very rapidly, out of all proportion to the size of the fœtus, and quickly extends from the internal os uteri to the apices of the horns. Not only is the embryonic sac co-equal in dimensions with the uterine cavity, but it becomes excessively long, and the tip of each cornu becomes necrotic. In some cases apparently the tip of the embryonic sac presenting at the internal os uteri also becomes necrotic.

The embryonic sac, developing co-equal with the size of the uterine cavity and firmly distended with the fœtal fluids, tends thereby to become securely fixed in its position.

Prior to the first conception, there exist in the mucosa of the uterus numerous slight elevations which are recognizable as the future maternal placentæ or cotyledons. When the animal is in estrum, these elevations become more prominent, are enlarged, and at the close of estrum there occurs from their surface a well marked hæmorrhage—menstruation.

When the embryo has attained a length of $\frac{1}{16}$ to $\frac{1}{8}$ in. the cervical canal becomes sealed by a gelatinoid mass, which we designate the uterine seal, and the uterine cavity becomes a a sealed sac. When the embryo is but $\frac{1}{4}$ in. in length, or less, there appear upon the exterior of the chorion distinct vascular areas, which are more or less thickened, and which correspond to the primitive cotyledons or placentæ in the uterine mucosa. These fœtal cotyledons or placentæ lie in intimate contact with those of the uterus, and correspond accurately with their location and area.

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If a cow aborts before the end of the fourth, and largely during the fifth month, there is no retained placenta. Very largely in early abortions the entire fœtal sac is expelled still surrounding the fœtus. As a result it is a common observation of breeders that abortion at this period is not as serious as when occurring later. The uterus promptly cleans up, and the animal as a general rule again conceives readily and carries the fœtus with reasonable security.

As pregnancy advances the intimacy of contact between the uterus and chorion continues to increase up to the day of parturition. If the uterus of a healthy cow in the last stage of pregnancy is opened immediately after slaughter the contact between the uterus and chorion is found to be very intimate, and a considerable degree of force is required to separate the two structures. They do, however, separate completely (see fig. 1).

The force which maintains the contact between the chorion and the uterus is unknown.

As we have already stated, the fœtal sac completely fills the uterine cavity, so that the close contact between the uterus and chorion is maintained mechanically by the internal pressure of the fœtus and its fluids. From without, the abdominal walls and viscera maintain an exterior pressure, so that the contact between the parts is further assured. This mechanical pressure, however, is not all.

There develop in the cotyledons of the uterus deep and intricately branched cavities or crypts opening towards the fœtal sac. Upon the surface of the corresponding vascular area of the chorion, the fœtal placentæ or cotyledons, there grow out numerous chorionic tufts (see fig. 2) consisting essentially of profusely branched capillaries with thin walls. These extend deeply into the crypts of the uterine cotyledons, and bring about the intimacy of contact necessary for the exchange of nutritive and waste substances and add to the security of the fœtal sac in the uterus. There is, however, at no time any continuity of tissue between the fœtus and the mother. The extremely thin walls of the maternal and fœtal capillaries come in immediate contact. They do not fuse, however, or in the ordinary sense adhere to each other, but remain throughout the life of the fœtus wholly separate.

Through the walls of these capillaries, lying in intimate con-

tact, the nutritive substances pass to and fro between the mother and the fœtus. No solid substance recognizable by vision has yet been known to pass through from the one set of capillaries to the other. Red blood cells do not pass and, so far as known, so long as the capillary walls remain intact no organisms visible under the microscope pass.

The force which maintains the contact between the fœtus and the uterus seems to be purely one of mutual attraction. So long as the fœtus lives and the navel cord remains intact, there appears to be a powerful attraction between the placentæ which we cannot define. When the life of the fœtus ceases, when the fœtal heart ceases to beat, or when the navel cord is ruptured, the attraction existing between the uterus and the fœtus ceases abruptly. The life of the fœtal membranes depends entirely upon the physiologic operation of the fœtal heart. The instant the navel cord ruptures or the fœtal heart ceases to beat, the placenta ceases to exist as a living tissue. The rupture of the navel cord causes an immediate emptying of the chorionic capillaries of blood, so that not only has the atraction between the two tissues suddenly ceased, but the volume of the fœtal capillaries has become enormously decreased.

It has been stated that the separation of the chorion is brought about by the contraction of the uterus. Histologists, however, tell us that there is no power in the tissues of the uterine placentæ to expel the tufts of the chorion. There are no muscle fibres in these parts, nor is there any elastic tissue of moment, so that they cannot in themselves contract and expel the chorionic tufts. It is not the mechanical compression of the uterine walls upon the chorion which detaches it. It is perfectly true that the uterus contracts promptly, and equally true that the contraction of the uterus expels the detached chorion from the uterine cavity, but it does not detach the placentæ. We must accordingly keep separated in our minds the dehiscence of the placentæ and the expulsion of the fœtal membranes from the uterine cavity. We occasionally observe dehiscence or separation of the placentæ without their expulsion.

The Nature of Retained Placenta.—When the normal detachment of the fœtal membranes is interrupted, we designate the phenomenon as "retained placenta." The exact nature of retained placenta has not been as well studied as it deserves. In extensive investigations upon the killing floors of abattoirs, in

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which 1,700 pregnant females were examined, diseases of the placenta other than septic inflammation were exceedingly rare. We observed in one case an intra-placental hæmorrhage, which would rather tend to detach than to detain the fœtal membranes. In another case we found a very extensive calcification of the cotyledons, which, had the cow been allowed to go her full term, would probably have caused serious retention of the fœtal membranes. We examined also an equal number of cows and heifers which were not pregnant, and in a number of these retained placenta existed. Except in the one case which we have related, the only cause of retained placenta observed was metritis, or rather cotyledonitis. Infection having entered the uterus, the cotyledons had become inflamed, swollen, and greatly enlarged. The swelling caused by the inflammation compressed and incarcerated the tufts of the chorion. There was constantly no inflammatory adhesion between the chorion and the uterus, but an incarceration due to swelling of the cotyledons.

We habitually hear it said that retained placenta in the cow is liable to be followed by metritis. This is a gross misconception of the facts. In all cases retained placenta is preceded by metritis. In other words, we should regard retained placenta not as a separate affection, but as a complication of metritis. The metritis must originate some time prior to the expulsion of the fœtus and have had time to develop, prior to calving or abortion, a sufficiently intense inflammation of the uterine cotyledons to incarcerate the tufts of the chorion. It has been erroneously assumed by writers that improper care of the cow after parturition, permitting her to take cold, or allowing her an improper diet, induces retained placenta. We commonly hear it said that general debility of the animal or prolonged labour causes retained placenta. In the very nature of the case this cannot be. The metritis which leads to the retained placenta may indeed cause great debility or critical illness of the animal, and equally it may cause difficult labour, because the metritis stops the normal contraction of the uterus. That the metritis may develop and increase in seriousness subsequent to abortion or parturition because of the retained membranes is perfectly logical, and this increased severity of the disease is too often mistaken for the beginning of it.

Retained placenta is the result of an infection which has entered the uterus. If we study retained placenta carefully we

find that clinically the retention originates about the internal os uteri and extends amongst the cotyledons toward the apices of the horns, chiefly in the gravid horn. A careful clinical study of a case of retained placenta usually shows that the cotyledons nearest the internal os uteri have become necrotic and sloughed off, or they have suppurated and the chorionic tufts have been forced out, or the chorion has otherwise become detached. Reaching forward toward the apex of the horn, it is found that the first cotyledons encountered are somewhat readily separated from the chorion because there has been a suppuration in the crypts of the cotyledons which has forced out to a great extent the tufts, or we find that the cotyledons rave become necrotic and drop off in the hand upon slight tension. Passing yet further toward the apex of the uterus, we find the cotyledons enormously enlarged and hard. They are not necrotic, and the chorion is difficult or impossible of detachment from the cotyledon. This suggests to us that the infection has entered the uterine cavity through the cervical canal and has slowly spread forward toward the apex of the cornu.

We have stated above that when conception has taken place and the embryo has reached a length of $\frac{1}{8}$ in. to $\frac{1}{4}$ in. the cervical canal becomes sealed and invasion of the utero-chorionic space apparently completely barred. It must therefore be that in some way the uterine seal has been overcome or the infection has occurred in spite of it. In our examination of 1,700 pregnant uteri we failed to find metritis in any case where the uterine seal was intact. Among the cases with sealed uteri was found an extensive infection of contagious abortion in numerous cases. The exudate, always massed about the internal os uteri, was frequently thickly distributed over the entire space between the uterus and the chorion. Yet, so long as the seal remained intact, metritis was absent.

The question consequently arises, "When and how does the infection which causes retained placenta invade the uterus and establish metritis?" In all our investigations upon pregnant animals we have not yet observed a case where any solution or disturbance of the uterine seal appeared to occur as a result of infection from the vaginal end. We have observed severe vaginitis with extensive collections of pus in the vagina, the bulk of it lying against the vaginal end of the uterine seal. Yet the

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uterine seal seemed resistant and to constitute a thoroughly efficient barrier against bacterial invasion.

On the other hand, in some cases of pregnant uteri containing the abortion exudate there were distinct evidences of solution of the uterine seal beginning at the anterior end. This would at least suggest to us that the uterine seal, when dissolved, is usually if not always accomplished by the action of bacteria which have been enclosed within the uterine cavity of bacteria when the uterus was sealed, or in some as yet unexplained manner has later invaded the sealed cavity.

We have elsewhere proposed the hypothesis and presented certain arguments to show that infection may enter the uterus prior to conception, but so long as the uterus remains sealed metritis does not occur. In some cases where the abortion exudate was present in the uterus early in pregnancy the uterine seal was absent and conditions suggested that in embryonic or early abortion the presence of the infection prevents the sealing of the uterus at all. It would consequently appear that in all probability retained placenta is due to an infection which has long existed within the utero-chorionic space and ultimately dissolves the uterine seal. A secondary invader may then enter the uterine cavity and induce metritis, or it may be that the organism already existing within the utero-chorionic space, once the seal has been destroyed, takes on a new character, in the presence of oxygen, to cause the metritis.

So far as we know, there is but one organism which may exist indefinitely in the sealed uterine cavity of the cow—the abortion organism of Bang.

It has long been recognized that there is an intimate relationship between contagious abortion and retained placenta. It is well known that abortion is very largely accompanied by retained placenta. In our Ambulatory Clinic for 1912-13, we attended thirty cases of retained placenta, including 27 cows, 2 mares, and 1 ewe. Of these definite record was made of abortion in the 2 mares, the 1 ewe and in 7, or 25 per cent., of the 27 cows. Others probably followed abortion and no record was made. How many of the remainder accompanied premature births due to the infection of contagious abortion or how many of those calving at full term had abortion exudate in the utero-chorionic space was not determined.

If the abortion occurs prior to the fourth or fifth month the fœtus is generally expelled enclosed in its membranes, or the membranes follow very promptly, and retention does not occur. Whenever abortion occurs later during pregnancy retention of the membranes is a very common occurrence. The probability

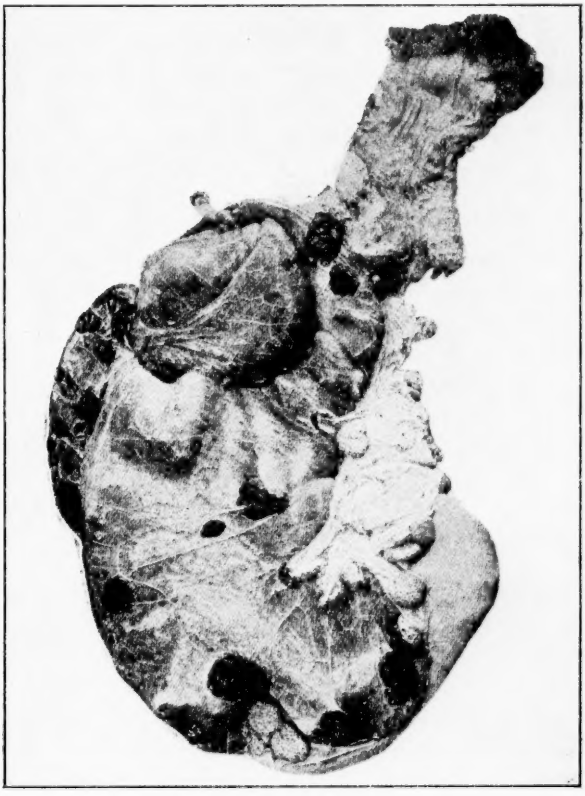

FIG. 1.

Uterus of cow in advanced pregnancy, opened longitudinally, showing fœtus (through chorion) in situ, some cotyledons detached.

of retention increases as the period of pregnancy advances. Retained placenta is more frequent in cases of premature birth than in those of abortion, because the premature births come at a later epoch in pregnancy with more elaborate chorionic tufts.

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In obstetric work we not infrequently meet with cases at full term where large volumes of abortion exudate are expelled prior to or during the expulsion of the fœtus. In such cases retained placenta is highly probable.

In our clinic we have made some effort to test the connection between retained placenta and abortion through the medium of the agglutination test applied to the blood of the affected cows.

An important element of uncertainty enters into the question of the relationship between abortion infection and retained placenta by the fact that, so far as we know, the abortion infection can only lead to retained placenta when it exists in large amount in the utero-chorionic space. It has been shown that the bacilli live in the udder, and we believe also that they live and multiply in the vagina and alimentary canal. No reason is known why the bacilli growing in these or other organs may not as readily cause the blood to agglutinate as do those growing in the uterus.

If we approach the question from another angle, we get some further light upon the cause, although still not conclusive. So far as the recorded investigations go, each and every cow having retained placenta which has been killed within a few hours after abortion or calving has uniformly shown in the utero-chorionic space an abundance of the abortion exudate containing the bacillus of Bang.

The number, however, is pitiably small. It is not safe, in the present state of our knowledge, to say that retained placenta is always the result of the infection of contagious abortion in the utero-chorionic space. Certainly an overwhelming majority of cases are referable to this cause, and it has not been clearly proven that any case of retained placenta in the cow has ever occurred in the absence of the infection of contagious abortion. If we could eliminate the infection of contagious abortion from the uterine cavities of pregnant cows we have every reason to assume that retained placenta would become as rare in cows as it is in other domestic animals.

The symptoms of retained placenta in the cow are usually so prominent that they need no description. In some cases the symptoms are exceedingly vague. Sometimes the retained placenta is either fragmentary or complete without any portion of the membranes showing externally. In one case, for example, we

observed, following abortion, the entire mass of fœtal membranes enclosed within the uterus. The cotyledons had all sloughed away from the uterus and remained attached to the fœtal membranes. There were no exterior evidences of the retention except the general appearance of ill-health and the discharge from the vulva. Fragmentary retention of the placenta toward the apex of the horn is quite common, and although the metritis which has caused the retention is fully recognized, fragments of the membranes frequently go unrecognized even until the death of

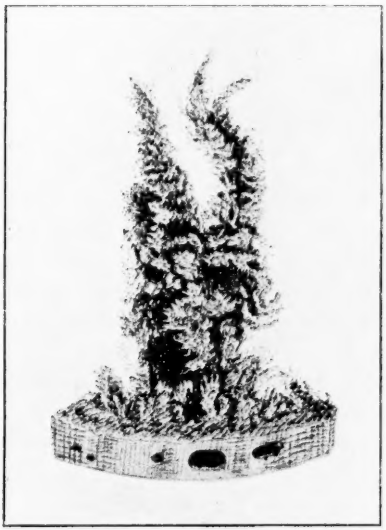

FIG. 2.

Chorionic tufts. (After Pomayer.)

the animal, and are revealed only upon *post-mortem* examination. They are so far removed from the cervical canal that they cannot be reached by the operator's hand, or perhaps they go unsuspected and are not searched for. Hence, in such cases, the symptoms are merely those of metritis, and the recognition of the retained placenta is possible only by careful manual exploration.

The handling of retained placenta is one of the most complex problems facing the veterinarian. It is a many-sided question.

The retention of the placenta intensifies the metritis which has caused it. The disease may have a fatal termination. It may cause a chronic metritis with pyometra, which interferes with the milk flow, and the pus constantly escaping from the vulva to some degree eventually finds its way into the milk, contaminating it and rendering it more or less dangerous for human use. A very large percentage of animals which suffer from retained placenta are afterward sterile and become a complete loss for breeding and dairying purposes.

In our "Veterinary Obstetrics" we have suggested three plans of handling:—

(1) The manual removal of the fœtal membranes. This should always be done whenever the chorionic tufts can be withdrawn from the crypts in the cotyledon. That is, whenever the fœtal membranes can be completely detached from the uterus this should be done. If, however, it is attempted to detach the membranes, and instead of the chorionic tufts pulling out from the crypts, the fœtal cotyledon tears away from the surrounding portions of the chorion and remains attached to the uterine cotyledon, nothing has been gained. Indeed, much has been lost. There is no longer that weight of the membranes dragging upon the now isolated membrane, which would tend gradually to detach the tufts.

If in using force to detach the fœtal from the maternal placenta the maternal cotyledon is torn or its neck is ruptured little good can come from the process. Rather, it is decidedly dangerous.

One of the great difficulties in the manual removal of the placenta is that, owing to the paralysed condition of the uterus, it has not undergone involution, and the most firmly retained cotyledons at the apex of the gravid horn cannot be reached by the operator. Were it not for the metritis which has caused the retained placenta the uterus within five or six hours after parturition would be reduced to such size that the operator could readily reach the apex of the horn, but it is not in these cases that the retention occurs.

When the manual removal of the afterbirth has succeeded the uterus should be well douched with a mild antiseptic and the fluid carefully drawn off. After this has been done, it is a good practice to deposit in the uterine cavity about ½ oz. of powdered iodoform enclosed in a gelatine capsule. This dissolves slowly, does

not irritate the uterus, and remains in the uterine cavity from four to six days, largely preventing decomposition and the extension of the infecting process.

(2) The expectant treatment, by which time is given for the spontaneous detachment of the membranes. The membranes may become detached in a variety of ways. As in all other inflammations, so in the inflammation of the uterine cotyledon, the disease process may abate, the swelling disappear, and as a result the incarceration of the tufts of the chorion cease, so that the membranes fall away spontaneously. Again, the crypts in the maternal placenta may undergo suppuration or a serous effusion may occur which may force the chorionic tufts out of the crypts. The cotyledons may become necrotic and slough off with the chorionic tufts still imprisoned. During this waiting period it has been proposed that the uterus be flushed out with antiseptic liquids. This is not very efficient. The disinfecting solution does not go into the uterine cavity, but into the fœtal cavity of the membranes, and does not come in contact with the uterus at all, except at those points where the membranes have become detached. Elsewhere the chorion lies in close contact with the uterine wall, attached here and there by the cotyledons acting as so many pillars crossing the utero-chorionic space from uterus to chorion. Between these two sacs it is not practicable to introduce a disinfectant, though possibly more might be done than has yet been attempted, especially if a soft rubber tube were carried along against the roof of the uterus above the roof of the chorion and gently insinuated between the two walls as far as practicable.

While this plan of douching the uterus with disinfectants is highly recommended by some, it is as thoroughly condemned by others. In a measure each position is correct. One of the great objections to introducing large volumes of fluid into the uterine cavity or into the cavity of the fœtal membranes which are lying within the uterus is that the paralysed uterus is not competent to expel the fluids introduced. If they are retained, their disinfecting value quickly ceases, and the fluids encourage rather than discourage the extension of the infection. The weight, too, of the fluids remaining in the uterus interferes very seriously with the process of repair, and is consequently highly objectionable. Again, if we attempt to force fluid into the uterus when the cervical canal is quite narrowed, and if the tube almost or

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completely fills the canal, the uterus may be actually ruptured, especially when the fluid is forced in with a pump.

It is difficult to remove the fluid when the uterus is paralysed. It will not syphon out with an ordinary tube because the membranes immediately occlude the opening. The cervix of the uterus is usually not involved, but contracts promptly, while the walls of the uterus itself, being inflamed, are paretic, so that they cannot contract properly, and hence the fluid is imprisoned. If we inject fluid, we must accordingly remove it if it is not promptly expelled. If the uterus still has sufficient contractile power to expel the fluid, this washes away much of the decomposing matter. If the veterinarian can remove the fluid by any means he will usually get good results from the douching. With some form of douching tube one may in many cases drain the fluid off fairly well. This may be aided by manual pressure per rectum. After the removal of the injected fluid we find that the introduction of ½ oz. of powdered iodoform in a gelatine capsule renders important service in preventing decomposition and checking the sepsis. Daily examination should be made, and as soon as practicable the membranes should be manually detached and removed.

(3) The manual removal of the placenta along with the uterine cotyledons. This plan has only a limited application. When the cotyledons have already sloughed away from the uterus and lie free within the uterine cavity or still attached to the fœtal membranes, removal is evidently the only logical course to pursue. In a few cases the cotyledons may be recognized by touch as being necrotic, enormously enlarged and hardened and very readily detached by slight tension, without being followed by any hæmorrhage. In such case the operator should not hesitate to remove the cotyledons entirely.

When all the cotyledons have sloughed away or been detached they do not reform, but in their stead in the next pregnancy the placenta takes the diffuse form like that of the mare. In old cows this diffuse form of placenta is quite common around the internal os uteri, indicating that during some previous pregnancy infection had been present in this region, which caused necrosis of the cotyledons. In a few cases examined by us upon the killing floor the placenta was diffuse throughout the entire extent of the uterus, indicating that as a result of serious infection at some previous time all the cotyledons of the uterus had been destroyed.

After the fœtal membranes have been removed or expelled under any method of handling, the veterinarian should see that the case is followed closely until the basic metritis in the uterus is overcome. It is not good practice to remove the placenta from a cow and not see her again and have no report regarding her condition. Whenever an animal is valuable, as in pedigreed stock, it is essential that the veterinarian should make frequent visits until the metritis is under complete control and no danger remains that the case will degenerate into one of chronic metritis or pyometra.

At best, the handling of retained placenta is merely an amelioration. The prognosis is by no means as good as many people believe. The case requires very close attention, and the results, especially in relation to breeding, are frequently very disappointing.

The prevention of retained placenta is a problem of far greater importance, which has not as yet been seriously attacked. From what we have stated above, it naturally follows that if we would prevent the metritis which causes retained placenta we must first of all prevent the infection of contagious abortion in the utero-chorionic space. It seems to us quite certain that if this infection could be eliminated there would be no more complaint of retained placenta in the cow than in other domestic animals.

If we accept the hypothesis which has been proposed by us, that the infection of contagious abortion enters the uterine cavity by the cervical canal prior to the sealing of the uterus, then the prevention of retained placenta must antedate conception. If we accept the more common hypothesis, that the infection of contagious abortion may enter the utero-chorionic space through a variety of avenues and at any epoch during pregnancy, the prevention of the metritis which causes retained placenta, in so far as it may be due to the infection of contagious abortion, must again be based upon some efficient method for controlling the abortion infection. In order to be efficient the precautions must again begin prior to conception, but it becomes more essential to continue the battle during pregnancy. We have not yet found an entirely efficient method.

We find that when we are able to induce a breeder to enter upon a comprehensive plan of permanent disinfection and hygiene of the genital organs of breeding animals the volume of retained

placenta becomes markedly decreased. That is, when we apply conscientiously an efficient method of sexual hygiene we ameliorate in a marked degree that great group of phenomena due to the infection of contagious abortion. Any measure that we will take to prevent the metritis which causes retained placenta will likewise decrease, according to our observations, the percentage of abortion, of premature birth, and of sterility.—*Report of New York State Veterinary College*, 1913-1914.

TUBERCULOSIS IN PHEASANTS.

By E. M. PICKENS.

Department of Comparative Pathology, Bacteriology and Meat Inspection.

Owing to the relatively small number of domesticated pheasants the public in general is not very familiar with the diseases of this species. A careful study of the literature on the subject shows that tuberculosis is a common disease, especially where these birds are kept in large numbers. Very few references are found of cases of tuberculosis in the wild pheasant. This is partly due to the fact that a comparatively small number of them are killed, and also that the chances of infection are not nearly as great as in the case of the caged birds. This condition is due to the fact that the wild bird is not as much exposed to infection from droppings, feeding dishes, runs, &c.

In Germany, Bierbaum [2] describes an outbreak of this disease where, out of forty pheasants, five died within fourteen days after purchase. He gave a very brief description of the lesions found in two hens. His diagnosis was made by finding the presence of the organism on microscopical examination.

In Italy, Baldi [1] described the lesions in several birds brought to the Veterinary Clinic of Perugia. The birds came from a large property, where the mortality had been quite extensive. On *post-mortem* examination the liver was generally found much enlarged, with nodules of various dimensions, some of which contained caseous material. Microscopical examination of these nodules revealed the presence of numerous bacilli of avian tuberculosis.

In New Zealand, Drew [6] reported the presence of the disease in that country. He described the lesions found in several birds.

Sibley [12] and also Van Es and Schalk [13] state that the disease occurs in pheasants in America.

Fagundes [7], Freidberger and Fröhner [8] and Law [9] all make the statement that the disease occurs in pheasants, but do not cite specific cases. Law gives a brief description of the lesions found.

Salmon [11] also makes the statement that the disease occurs in pheasants. He quotes Nocard to the effect that this disease may be diagnosed in the living birds by means of the subcutaneous tuberculin test. He gives the dose of tuberculin of from 5 to 10 cg.

Morse [10] writes that tuberculosis is fairly common among domesticated pheasants and is often spoken of as "going light." He continues to say that all cases of "going light" are not

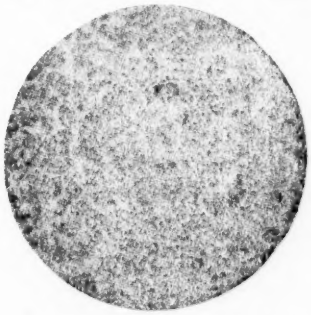

FIG. 1.—Small tubercle in liver of pheasant showing arrangement of giant and epithelioid cells. x 50.

tuberculosis. He gives a brief description of the *post-mortem* findings.

Cadiot, Gilbert and Roger [3 and 4] have done considerable work on this disease. They have infected pheasants with the avian tuberculosis organism and allowed them to die; proving that the avian organism will cause the disease in pheasants.

Cadiot [5] describes several epizootics of the disease among pheasants. He gives a careful description of the lesions found, and a comparison of the lesion of the pheasant and the fowl. He says that the tubercular lesions in the two species are practically indistinguishable to the naked eye. A histological examination, however, shows several differences. Some of the most important are: In the pheasant the tubercle bacillus causes a development of epithelioid cells; these soon undergo molecular degeneration, and are succeeded by the formation of an abundance of connec-

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tive tissue, which surrounds pseudo-vascular cavities, and undergoes hyaline necrobiosis, which, though at first limited to the

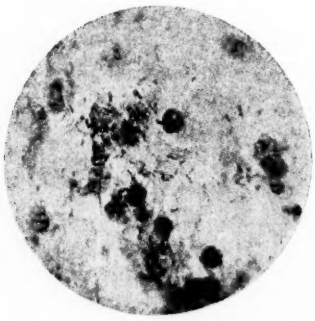

FIG. 2.—*Bacterium tuberculosis* in smear from spleen of pheasant. x 750.

centre of the tubercle, and producing the appearance of a clear centre surrounded by special epithelioid cells, afterwards extends to the whole of the new growth and tends to encystment.

HISTORY OF AN OUTBREAK IN NEW YORK STATE.

A male pheasant was received at the laboratory for examination in the early part of May, 1914. At this time the weather was particularly warm, and the senders had neglected to pack the specimen in ice, so on its arrival it was decomposed to such an extent that an examination was impossible. The senders were requested to ship us another affected bird properly packed, and also to give us a history of the trouble.

A short time afterward a second bird was received, together with a somewhat complete history of the outbreak. This was as follows: "For the past three years there had been a noticeable affection among the pheasants. The symptoms showed themselves in various ways, chiefly by loss of appetite and continued inactivity. Between 10 and 20 per cent. died each year. Most of the deaths occurred in the spring or early summer, the hens being more susceptible than the cocks. Death was preceded by loss of appetite, drooping of the wings, paleness about the head, and a listless and inactive appearance. These symptoms were noticed for about a month before the bird succumbed. About

two or three days prior to death the bird refused all food and showed little, if any, activity.

"The pheasants were housed in open wire cages, placed at the end of a field, and in the vicinity of a spruce hedge. The cages were oblong in shape and large enough to permit of some flying. The air drainage was good and the location was high enough to be free from any excessive dampness in the soil. Every spring, and once during the season, the cages were thoroughly limed and disinfected. Grass was sown each season, and the birds got a sufficient supply of green food. Water was supplied once a day, and the birds were fed a ration made up of peas, wheat, cracked corn, oats, and a little millet. It was noticed that the corn was consumed first, after which the ration was made to contain about one-third cracked corn. This season the cages were removed to fresh ground."

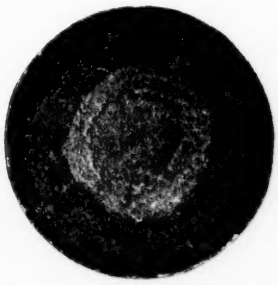

FIG. 3.—Small tubercle in liver of fowl showing arrangement of giant and epithelioid cells. x 50.

The case in hand was a female bird of average size, probably 2 years old. She was emaciated to such an extent that the pectoral muscles could scarcely be felt. The feathers were much ruffled and showed a general unthrifty condition. Upon *post-mortem* the following conditions were found:—

Liver.—The liver was about natural size, and was thickly studded with yellowish-white, irregularly round areas. These areas varied in size from being scarcely perceptible to the naked eye to about 1 cm. in diameter. All surfaces of the organ were affected alike. In the intervening spaces between these areas where the liver tissue could be seen, it was pale and seemed to have undergone parenchymatous degeneration.

Upon section the above mentioned irregular, light-coloured areas were found to extend below the surface of the organ, and were distributed more or less uniformly throughout. Upon close examination these areas showed what seemed to be caseation necrosis. The smaller areas were of a greyish-white colour, while the larger ones were somewhat darker. They were a dirty yellowish white, and appeared to be homogeneous, having a more or less hyaline appearance and a caseous consistency.

Spleen.—The spleen was about twice its normal size, and was so thickly studded with these yellowish white areas, that it was practically impossible to see any normal tissue. In certain places minute, light-coloured areas could be distinguished standing out against the darker splenic tissue, but almost universally these areas had grown to such a size that they had become confluent with their neighbours, and the whole organ gave the appearance of being a single, large yellowish-white nodule. Upon section, the organ showed the same condition as the liver, except that there was very little normal tissue to be seen. The interior of the organ appeared to be composed of the same caseous material, and was of the same consistency. No calcification was found. In the other organs of the body no marked lesions were found. The intestines, kidneys and ovaries, as far as could be determined. were practically normal.

Numerous smears were made from diseased areas of both the liver and spleen. These were stained for tubercle bacteria. Upon examination of these smears the characteristic acid-fast organisms were found in large numbers. They were long slender rods, resembling very closely, if not identical with, the avian tuberculosis organism.

After the smears were made the spleen was cut up into very small pieces and used for making cultures. Glycerine agar and egg media were employed. These cultures were incubated at 37° C. for several weeks. One pure culture of *Bacterium tuberculosis* was obtained from this material.

From the liver pieces were taken for sectioning from several different places. These pieces were fixed in Zenker's fluid, sectioned and stained. The stains used were hæmatoxylin and eosin, hæmatoxylin and picrofuchsin, and the Ziehl-Neelson stain.

Histological Examination of the Liver.—In certain areas the organ appeared practically normal, the cell bodies and nuclei

taking the stain well and standing out sharply, while in other areas small amounts of fibrin, together with cloudy swelling and fatty degeneration, were found. The tubercular areas were scattered irregularly throughout the tissue. These lesions varied in size from as large as two or three liver cells to an area covering several lobules. The structure of the lobules seemed to vary somewhat with their size. The smaller ones contained a centre made up of an irregular mass of giant cells. These cells were irregularly oval and varied somewhat in size. For the most part they showed a large, more or less homogeneous cell body, and many nuclei. The nuclei were sometimes collected in a mass in the centre of the cell, at other times they formed a ring, this not always being complete, and giving the appearance of a horse-shoe. At other times the nuclei were scattered irregularly through the cell. Surrounding this mass of giant cells could be found a rather thin connective tissue stroma, which contained a large number of small round cells, that appeared to be lymphocytes. This zone appeared to come into direct contact with the normal liver tissue. In lesions of this size the line of demarcation seemed to be very sharp between the normal and the pathological tissue. The different zones found in the avian tubercle could not be made out.

In some of the larger lesions a necrotic centre could be seen. This was surrounded by an irregular zone of giant and epithelioid cells. This zone was surrounded with a comparatively wider one made up of connective tissue stroma, infiltrated with large numbers of lymphocytes and polymorphonuclear leucocytes. This zone was circumscribed by a very narrow reactionary band consisting largely of lymphocytes. In these lesions, as in the above, the line of demarcation between the normal and pathological tissue was very sharp.

The largest tubercles, like the ones just described, contained a necrotic centre. In places parts of this had either become disintegrated or had dropped out while being sectioned, leaving vacant spaces. Scattered irregularly through this zone could be seen numerous giant cells, which had undergone more or less degeneration. Those nearer the centre showed a greater amount of degeneration than those nearer its edge. This zone gradually radiated out into another zone made up of connective tissue stroma containing large numbers of lymphocytes and polymorphonuclear leucocytes. This zone contained many giant cells scat-

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tered irregularly throughout. The outer border of this zone seemed to come in direct contact with the normal tissue of the liver, so that, as in the above cases, the line of demarcation between the normal and the abnormal tissue was very sharp.

The examination of the specimens stained with the Ziehl-Neelson stain for the causative organism showed many tubercle bacteria. A few of these organisms could be distinguished scattered through the necrotic centre, but the organism occurred in large numbers in the zone made up of the connective tissue stroma and lymphocytes. This was especially true at the border of the lesion adjacent to the normal tissue.

The owner of the birds was informed of our findings, but he was unable to enlighten us in any way as to the source of the infection. He was advised to slaughter all birds showing excessive emaciation and to thoroughly disinfect the cages. This process should be repeated at short intervals as long as any signs of the disease could be found. In this way it was hoped to eventually eradicate the disease without destroying the entire flock.

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—From *Report of New York State Veterinary College*, 1913-1914.

Clinical Articles.

AN UNDIAGNOSED DISEASE.

By G. MAYALL, M.R.C.V.S.

I ATTENDED a bay mare cob in July for a sore on the off lip of her vulva and sores on her udder. These were treated with wound oils and antiseptic dusting powder, and although troublesome to remedy had apparently healed in about three weeks' time. The mare was then turned out to grass and was brought up again in the middle of September. I was then sent for to examine her. I found her covered with scabs from head to foot. One side of her vulva was deformed and eroded and her teats dropped off from the udder. Scrapings from the skin revealed purulent discharge underneath the scabs; a powerful lens and examination by Pugh's mange tray method revealed no acari. The mare was a brute to do anything with and two twitches had to be put on her to obtain any scrapings. Owing to the difficulty of any proper treatment, to avoid danger of infection (supposing there was any) and to safeguard against any accident to man incurred in treatment, the owners had the mare shot. Was the disease necrobacillosis? If so, it is the first generalized case I have ever seen.

SPINDLE-CELLED SARCOMA ON INSIDE OF CHEEK OF COLLIE DOG.

By CRITTENDEN ROSS, D.V.M.
New York, N.Y.

ANSWERING a call to see a collie dog on June 3 that was reported unable to eat, we found, upon examination, imbedded in the cheek a large tumorous growth which appeared necrotic and gave off a very offensive odour. At the owner's request the patient was treated but the client warned of the seriousness of the condition. Four days later, at the solicitation of the owner, the patient was operated upon. All efforts were used to remove the entire growth and both upper and lower maxillæ were curetted. In the centre of the growth was found a small tooth, resembling a deciduous incisor. Samples of this mass were sent to a local laboratory, also to Dr. B. F. Kaupp, of West Raleigh,

N.C. The former laboratory was quite busy at the time, and as the material was sent in the fresh state, only a report from gross examination was made, which stated that the material did not appear as if from a malignant growth, and that once thoroughly removed would not be apt to reappear. As a result of this report a second operation was performed, as the growth had returned in ten days. Shortly after this operation sufficient time had elapsed to receive word from Dr. Kaupp, which bore the information that the growth was a large spindle-celled sarcoma; but time was allowed to see the outcome before destroying the patient, which resulted in the return of the growth after a period of only eighteen days. The patient was then destroyed.—*American Veterinary Review.*

FOLLICULAR MANGE.

By H. S. EAKINS, D.V.S.

Professor of Histology and Materia Medica, Division of Veterinary Medicine, Colorado Agricultural College.

Various authorities state that treatment of follicular mange in dogs is most unsatisfactory. As I noticed another item in this connection in the August number of the *Review*, I thought the veterinary profession would be interested in the following case reports:—

White English terrier entered clinic March 16, 1915. Squamous type of follicular mange very extensive over head, including face and ears; extensive over neck and shoulder regions; scattered over back, legs, and under surfaces of body. Parasite demonstrated. Hair was shaved from head, neck and shoulder regions where the lesions were numerous. Hand treatment with bristle brush, using sapo mollis and 2 per cent. aqueous solution of liquor cresolis compositus was a preliminary measure. After skin become dry iodine petrogen 10 per cent. was thoroughly rubbed into the skin wherever lesions presented themselves. In order to prevent the spread of the condition to the few remaining healthy skin areas, there was applied to these areas oleoresina aspidii and balsam of Peru in linimentum saponis mollis. Daily treatment with the iodine petrogen resulted in a rapid recovery, and the condition did not spread, due no doubt to the daily treatment of the non-affected skin areas with the above outlined combination.

Imported Japanese spaniel entered clinic July 13, 1915. Squamous type of follicular mange very extensive over entire head, being worst around the eyes and on the ears, and extended to forelegs. Pruritus was intense and dog was in agony. The right cornea had been scratched, resulting in an opacity. The long hair was clipped and preliminary treatment as in first case was given. The nails were clipped and infants' stockings were securely placed on feet, this in an effort to prevent scratching. To the lesions was applied daily equal parts of oleoresina aspidii and iodine petrogen, 10 per cent., being rubbed in well. The dog became quiet. The worst lesions succumbed to the above treatment in about seven days. However, the mange had spread to the body, hind legs, and tail; but was quickly brought under control with the iodine petrogen male fern treatment. A thorough examination of the patient three weeks after entrance to the clinic did not reveal a single lesion of mange. During the course of treatment, nuclein (for human use) was given subcutaneously every two days. The opacity of the cornea was overcome through a 1 to 2,000 cyanide of mercury aqueous solution.—*Journ. Amer. Vet. Med. Assoc.*

MILK FEVER IN A COW.

By G. MAYALL, M.R.C.V.S.

On October 3 I was called to a horned, crossbred cow, 5 years old, which my *locum* had attended a year previously for milk fever and had treated successfully in the usual manner. On my arrival I found the animal conscious with her head extended, grinding her teeth, and unable to rise. Temperature 100, pulse 45, respirations 24. I gave a draught composed of acid hydrochlor. dil. 1 drachm, tr. nux vom. 1 drachm, tr. gentian 1½ drachms, liquor taraxacum 6 drachms, water to a pint. An enema given at the time brought away a lot of black, lumpy, bad-smelling excreta. A second bottle of medicine as above was left. The next morning the cow was comatose, head turned round to flank, stertorous breathing, temperature 102. I injected the udder with 2 drachms of pot. iodide in 2 pints of warm water (I always prepare the injection at home and take it with me ready for use), blowing in plenty of air afterwards, and tying the ends of the teats with small bandages. The udder was then massaged, the bladder and rectum emptied, and a similar draught administered to that previously given and one left. The black and lumpy condition of the fæces was still present.

The following day the cow appeared considerably better. She had been up in the morning, but was down when I saw her, but quite conscious, breathing rapidly and a temperature of 105° F. I had her back and sides rubbed with liniment, a cold pack put round her horns, and gave her a draught made up of liquor

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am. acet. ½ oz., pot. nit. 1 oz., sodæ hyposulph. ½ oz., water to 1 pint. A similar dose of medicine was left to be given at night. The bandages from the teats had been removed and the milk drawn. The black condition of the fæces still persisted, but the bowels were acting better.

Briefly, this cow continued to improve and on October 11 I ceased attending her and she has gone on all right.

The hurried breathing persisted, even to the last day I saw her, but the fæces had regained their normal colour and consistency, but as the bowels were not acting well on October 6 I gave a hypodermic injection of arecoline hydrobromide ½ gr., physostigmine benzoate ½ gr., strychnine sulphate ⅛ gr. At this farm I have had two milk fever cases lately, both of which have recovered, but they have been followed by rapid breathing persisting for several weeks after apparent recovery from milk fever. The dark, tarry condition and bad smell of the fæces I have seen repeatedly in both cows and sows after parturition. Some cases of milk fever, clinically at any rate, show pronounced symptoms of an auto-intoxication.

Presumably owing to the difficulty of obtaining chinosol many practitioners will return to the use of pot. iodide. It has always served me well, and although I have frequently used chinosol satisfactorily, yet I have never seen any adequate reason for discarding pot. iodide and I have used it off and on for about fifteen years.

A PREMATURE BIRTH.*

By A. L. DANFORTH, D.V.M.
Watertown, N.Y.

An interesting case came to my notice while at Goshen which I thought might be of interest to the readers of the *Cornell Veterinarian*. It was interesting to me in that I had never heard of so young a fœtus being born and still live. It occurred on the farm of one of our clients and we were called by this owner asking if we cared to come and see it. The man keeps a careful record of his breeding stock, and there is no reason whatever for doubting the authenticity of the facts given.

Grade Holstein, aged 6 years, third calf. Freshened March 29, 1913, going full time, was bred again on May 15, 1913, and on October 22, 1913, gave birth to a live bull calf weighing 19 lb. The cow having been bred 160 days previously. The calf was apparently normal in every respect except for size. He was kept until February 11, 1914, and sold, at which time he weighed 140 lb. He was by a three-year-old registered Holstein bull.

I am wondering if there are on record any younger calves which have lived.—*American Veterinary Review.*

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* Reprinted from the *Cornell Veterinarian*, July, 1915.

VIEWS TAKEN BY A BRITISH ARMY VETERINARY OFFICER AT THE FRONT.

On the retirement from Mons, August, 1914.

A shell demolished this house and killed four men who were playing cards.
Ypres, November, 1914.

A friendly tea-party at Soupir, on the Aisne, September, 1914.

A Rest Camp. Horses on the line on the advance to Mons.

Translations.

PREMATURE BIRTH AND NORMAL BIRTH IN THE COW DURING THE SAME GESTATION PERIOD.

By Dr. A. SALVISBERG.
Tavannes.

Professor Guillebeau recorded in this Journal for March two cases of premature birth and normal birth in the same animals. These cases are not only interesting to veterinary surgeons and breeders as exceptions to the rule, but they are important from a forensic point of view. A four-year-old Simmenthaler cow which had calved for the first time on August 20, 1912, was put to the bull on November 9 and December 30 in the same year. Afterwards no more heat was noted and the animal appeared pregnant. On April 23, 1913, I was called to examine the cow for a claw ailment. Whilst opening the digital abscess and bandaging the foot the animal struggled violently. Half-an-hour after this trifling operation the cow aborted a goose egg-sized fœtus. On May 5 and 26, 1913, the animal was in season, but was not covered. On June 16, being on heat again, she was sent to the bull. Afterwards œstrum ceased. During the month of July the quantity of milk decreased and on October 16, 1913, the cow gave birth to a live bull calf, 50 kilogrammes in weight. About an hour after the birth a normal afterbirth came away, and after ten minutes a second. Each placenta was complete with a well-developed umbilical cord and without any putrefaction. The size of both placentæ was equal.

Doubtless the cow conceived on December 30, 1912, and carried twins, one of which was aborted on April 23, 1913. The two fully developed placentæ appeared worthy of note to me as well as the three heat periods repeated at intervals of twenty-one days during the pregnancy.—*Swiss Journal of Veterinary Medicine.*

SOME CASES OF POISONING BY TAXUS BACCATA IN GOATS AND HORSES.

By HERMANN S. FRENKEL.
Pathological Institute, Utrecht.

In October, 1914, an owner reported the death of three goats and sent the carcase of one to the Institute for examination. At the same time two older goats were reported ill showing inappetence, groaning, discharge of white foam from the nostrils, great weakness and convulsions, and one died the following day. The animals had received grass, cabbage leaves, and fir needles. These latter led one to suspect poisoning by yew tree foliage. On section there was very advanced putrefaction. The intestines were tympanitic and there was diffuse hyperæmia of the

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mucous membrane of the intestine and abomasum. The rumen wall was very rotten and the chyme firmly glued to the mucosa. In the green contents of the rumen were found numerous, entire, equal-sized, 3 cm. long and 4 mm. broad, linear leaves. The reticulum, the omasum, the abomasum and the intestines were empty. The lungs showed intense œdema.

The author concludes these were cases of taxus poisoning. Rumination ceased immediately after taking this food, and the poisonous principle of yew leaves "taxin" was quickly taken up from the rumen and incorporated in the digestive juices.

A military horse which died in a few minutes furnished another case. Oats, chop, pieces of grass, entire and chewed yew leaves were found in the stomach on *post-mortem*. The blood was dark reddish brown and clotted badly. Examination for anthrax was negative. The leaves and pieces of leaves collected from the stomach weighed 200 milligrammes, which was a thousandth part of the lethal dose. Frenkel believes that the amount taken must have been greater than that found and that the juices of the stomach quickly changed the leaves and made them unrecognizable, and at the same time much of the stomach contents had escaped into the intestine. Two other military horses in the same yard had access to yew trees and one was found dead and the other died quickly, showing great prostration and a markedly slow pulse. Another military horse showed symptoms of great unrest, violent sweating, great weakness and decubitus. The excrement was dry and dark coloured, the breathing rattling and the breath stinking. Portions of food and slime were discharged through the nostrils. The horse was slaughtered and section gave rupture of the stomach. Taxus needles were found in the stomach contents.

The conclusions the author draws are partly contrary to those hitherto given on taxus poisoning. It appears that yew leaves mixed with other food and taken immediately after feeding may be unaccompanied by any violent symptoms and that the animal may slowly become accustomed to the taxus poison. Further, the occurrence of poisoning depends on the individual disposition. It appears that young, clear green coloured leaves and twigs of *Taxus baccata* are not poisonous, and that the toxicity depends on the oldness of the parts taken. In opposition to the conclusions of Romberg and Ehrenberg, Frenkel considers that goats are very susceptible to the poison.—*Swiss Journal of Veterinary Medicine*. G. M.

Reviews.

Anæsthesia and Narcosis of Animals and Birds. By Frederick T. G. Hobday, F.R.C.V.S., F.R.S.E. Demy octavo; pp. xii + 86; plates 5; figures in the text 19. Price 5s. net. Baillière, Tindall and Cox, 8, Henrietta Street, London, W.C.

It has often been written and said that all veterinary surgeons use anæsthetics nowadays, and yet this is the first book giving expert guidance on the subject. It will prove of inestimable value to the humane veterinary surgeon, to the practitioner who has used anæsthetics for some time, to the student, and to the man starting in practice who wishes to avoid some of the pitfalls due to an incomplete knowledge of the forces which he endeavours to summon to his own aid and to alleviate animal suffering. No man in our own profession could come to the task of writing about anæsthetics with a wider or more extensive experience of his subject than the author. With perfect trust and confidence, then, this little book may be recommended whole-heartedly to all those who wish to be in the front rank of the science and practice of their professions, and its pages ought to appeal to an even wider public than is comprised within our limited circle.

The work is dedicated to Professor Augustus Waller, M.D., F.R.S., Professor of Physiology in the University of London, "whose work on the administration of chloroform has done so much to demonstrate the safety of this anæsthetic when given by dosimetric methods." The Professor and Mr. W. L. Symes, M.R.C.S., have given the author every encouragement in his work, and Professor Wooldridge and Mr. Victor Fisher (Managing Editor of THE VETERINARY JOURNAL) have lent valuable aid.

The little volume is divided into eleven chapters, the greatest space being naturally taken up with general and local anæsthetics. There is an advanced and interesting chapter on intraspinal anæsthesia (confined almost entirely to the dog) which is well illustrated, whilst a short section devoted to narcotics is very acceptable. In this latter connection we should like to have read the author's opinion on the best and quickest narcotic for destroying the horse. Recently we were asked to put a favourite old cob to his final sleep, and we accomplished it by giving chloral by the mouth and rectum, but it took about 12 oz. of the drug to accomplish the end.

Guidance is given in plain language on the use of all the general and local anæsthetics, and antidotal treatment and treatment of the patient when recovering from the hypnotics are discussed. Chloroforming standing is explained, and those who advocate this method will benefit by reading the author's lines on the matter.

There is no branch or phase of the subject that has been overlooked. The information given is concise and complete. The illustrations are good. We are indebted to the writer for pioneer work in a field in which we, as humane and skilled men, must

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advance. No more opportune moment than the present could have occurred for the birth of a book which marks an epoch in the literary and scientific history of our profession. G. M.

The Annual Report of the Chief Veterinary Officer for 1914. Board of Agriculture and Fisheries. Publishers: Wymans and Sons, Limited, Fetter Lane, London, E.C. Price 3d.

Sir Stewart Stockman having been busily engaged in research into the treatment of swine fever by serum and vaccination the issue of this report has been somewhat delayed. Foot-and-mouth disease has been pretty prevalent, eleven outbreaks in the county of Lincolnshire in the district of Lindsey helping to swell the total considerably. Eight other cases in various parts of England occupied the attention of the officials, and all the outbreaks were successfully dealt with in the usual way.

There was an increase in the number of cases of swine fever, but the total pigs slaughtered by the Board was proportionately smaller than in previous years. A report has been prepared by the Chief on the treatment of the disease by serum and there is reason to believe that great use will in future be made of the method in the control and eradication of the epidemic.

Glanders shows a decrease due chiefly to a wide recognition of the use of mallein in the extermination of the disease. A word of caution is given as to the acceptance of certificates of freedom from glanders when purchasing new ponies for the pits. A certificate brought with a pony to be sold may not apply to the animal or be of too ancient date to be valuable. Russian ponies were, as usual, the chief hosts of infection, eleven out of eighteen of the cases concerned them.

Only 722 outbreaks of anthrax were confirmed out of 1,150 suspected cases investigated. A dog and two cats died from eating the flesh of anthraxed carcases, and some animals bedded on shoddy caught the complaint; whilst two horses, the one carting hides and the other dragging an anthraxed cow carcase, died from the malady.

Sheep scab shows a decrease and tuberculosis in swine an increase. From cases encountered at the abattoirs in Glasgow and Birmingham and in making investigations into suspected swine fever cases it is assumed that probably 3 per cent. of swine may be affected with tuberculosis, so that measures to prevent the occurrence of the disease are worthy of notice.

The Tuberculosis and Mange Orders have been suspended during the War, and no very enlightening information is obtainable about them. 2,892 cases of swine erysipelas were encountered. New laboratory premises are now being constructed by the Board and a supply of serum and vaccine for swine erysipelas will soon be available. The manufacture of anti-swine fever serum has already been begun by the Board, and up to the end of December 137,000 c.c. had been distributed for use.

Redwater and epizootic abortion were the subjects of investigation by the Board. Experiments with vaccine in the latter disease have given encouraging results.

A full report in connection with the results obtained with anti-swine fever treatment in practice is promised.

There is evidence of gratifying advance in the activities of the Board and the benefit that stockowners and the general public are getting from the work of this State Department deserves to be appreciated.

ARMY VETERINARY SERVICE.

Army Veterinary Corps.

Appointment of W. F. Morton to be temporary Lieutenant; temporary Lieutenant W. F. Morton to be temporary Captain; G. Whitehead to be temporary Lieutenant; Lieutenant C. A. A. Ewin to be Captain; Lieutenant H. McVean to be Captain; Lieutenant A. Scotson relinquishes his commission on account of ill-health; Temporary Lieutenant R. J. Collings to be temporary Captain; temporary Lieutenant P. A. Wilks relinquishes his commission; Temporary Major E. E. Bennett to be temporary Lieutenant-Colonel whilst holding appointment of Assistant Director of Veterinary Services. Captain J. O. Andrews to be temporary Major whilst holding appointment of Assistant Director of Veterinary Services. Temporary Lieutenants to be temporary Captains: A. Young, W. D. Halfhead, J. McL. Dawson, E. A. Phipps, A. R. Smythe, E. S. Dixson, J. S. Young, W. W. Lang, E. E. Jelbart, D. Hannay, A. C. Smart, J. B. Walker, R. C. Allinson, J. E. Young, J. Godber, R. P. Johns, J. P. Dunphy. The following to be temporary Lieutenants: A. Plant, W. Gardner.

Lieutenant H. Chown, from S.R., to be Lieutenant; temporary Lieutenant W. Scott to be temporary Captain.

Temporary Captain F. C. Gavin to be temporary Major, while holding appointment of Assistant Director of Veterinary Service; temporary Lieutenant L. S. Sedgwick relinquishes commission on ceasing to be employed.

W. F. Wright to be temporary Lieutenant.

J. R. Welsby to be Lieutenant.

Lieutenant G. McIntyre to be Captain.

Temporary Lieutenants to be temporary Captains: E. Nicholson, W. H. Priston, C. W. Makinson. Canadian veterinary surgeons to be temporary Lieutenants: J. C. Miller, W. F. Macdougall, J. W. Welch, T. W. McMahon, J. R. C. Andrews.

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Temporary Hon. Lieutenant E. G. R. Fairholme to be a Deputy Assistant Director of Veterinary Services (graded for purposes of pay as a Staff Captain), and to be temporary Captain whilst so employed.

Temporary Lieutenants to be temporary Captains: R. T. Davis, J. Hill, T. Hogg, R. J. Little, H. J. Allen; J. Purdy to be temporary Captain; W. F. Llanagan to be temporary Lieutenant; W. H. Mawdsley to be temporary Quartermaster with hon. rank of Lieutenant.

Temporary Lieutenants to be temporary Captains: J. M. Smith, J. Pugh. Temporary Lieutenant F. C. Gillard relinquishes his commission on the termination of his engagement; temporary Lieutenant P. A. Robinson relinquishes his commission.

W. H. Williamson to be Lieutenant.

Temporary Lieutenant J. Pollard to be temporary Captain.

OBITUARY.

William Henry Jones, M.R.C.V.S., of Chatham, died on May 28, at San Francisco, California, U.S.A., from rheumatic complications. Graduated July 1, 1875, London.

Veterinary-Major Gerald Herbert Fenton, late Army Veterinary Corps. Died on October 2, 1915, aged 63. Graduated April 16, 1874, N.Ed.

Captain John Storie, A.V.C. (T.F.), East Linton, Prestonkirk, Haddingtonshire. Died on October 4, 1915, aged 59. Graduated April 10, 1875-1880, N.Ed.

Note.—All communications should be addressed to 8, Henrietta Street, Covent Garden, London, W.C. Telephone, 4646 Gerrard. Telegrams, "Baillière, London."

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